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A Clinical Lecture Delivered at the Medical Department of the University of Denver.

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PROFESSOR OF DISEASES OF CHILDREN AND CLINICAL LECTURER IN THE UNIVERSITY OF DENVER. PATHOLOGIST TO SAINT LUKE'S HOSPITAL.

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Gentlemen:—We have studied pulmonary tuberculosis again and again, but most of the cases which you have seen have been of comparative recent origin. We have to-day a series of four cases of what might be called common chronic phthisis in which the origin dates back ten to twenty years. Such cases for the purpose of study are very interesting and instructive and from such cases we can learn how resistant the body cell is often to the tubercular germ. To study any one case in a pathological way would be to extend our time almost indefinitely. What a vast array of pathological changes must take place in these chronic cases of pulmonary tuberculosis. Think for a moment what such an investigation would lead us into considering; the various forms of pneumonia,—croupous, catarrhal and interstitial, the deposition and formation of the tubercular follicle and nodule, the breaking down or caseation of this mass, the formation of cavities, the subject of adhesive pleurisy, bronchitis, tubercular involvement of the bronchial glands, compensatory emphysema and compensatory trouble in the heart. What an interesting field of study!

* * *

J. K. M., 39, Louisiana, brass finisher, single, came to Colorado November 11th, 1892. The family history is unimportant. There is no history of consumption in any member.

Patient had all of the diseases of childhood, including a severe attack of whooping cough. While in his teens he enjoyed excellent health. At twenty he began having malarial fever and for two years he suffered with it. During this time he had considerable jaundice. From 1878 to 1883 he had good health.

In January, 1883, twelve years ago, while lying in bed, patient having just retired, he suddenly turned on his right side and with a cough a large amount of blood came up from the lungs. For two hours blood came up in quantities. He says he certainly spit up fully a quart of blood. He was in bed for weeks and every week

for a month or two he would cough up a little blood. Then for eighteen month he enjoyed fair health, had but little cough, had no haemorrhage. Then he began to bleed again and on an average he had a pulmonary haemorrhage once a month until 1887. In June of this year he had a profuse loss of blood. For five hours he bled continuously. Was quite prostrated after it. Between the haemorrhages he had a slight cough, with a few dyspeptic symptoms and some pain over the spleen.

Since 1887 he has had no haemorrhage and has enjoyed good health with the exception of some dyspepsia, which gave way before pepsin and carbolic acid. He denies venereal disease. One year ago he began to have a pronounced morning cough and many a time he coughed for an hour. He would expectorate white, thick, tough, sticky mucus. After getting to work his cough would leave him until the following morning. For a whole year this has continued. Patient came to our clinic five days ago. He says his general health during the past year has been good, the appetite is good, the bowels are regular and he has had no night sweating. He has held his usual weight and besides a little pain in the upper part of the chest and in right hepatic region, he has been well, with the exception of his cough. The pulse and temperature are normal.

The physical examination of his chest shows an expansion of one and one-half inches, with much fullness of the chest in the lower half. On auscultation there is a constant and evident diminution of the respiratory murmur below the inner half of the left clavicle. Over the extreme apex of the left lung there is prolonged expiration and a few slight semi-dry rales. The right apex gives a rough prolonged expiratory murmur. The breath sounds are weak all over chest. The examination of his sputa shows a few bacilli of tubercle.

Here we have an interesting case in which the progress of the tubercular lesion has certainly been very slow. Twelve years he suddenly had a pulmonary haemorrhage and for four years he had a great many such haemorrhages.

I am asked how I know these haemorrhages to have been pulmonary. I do not know, but I believe them to have been so, because they were accompanied with a cough, in a case where the heart was normal, where there was no pronounced gastric symptoms and because they appeared at such infrequent intervals.

It is sometimes a difficult matter to differentiate between a haematemesis and a haemoptysis, long after the occurrence of the haemorrhage. We must remember that blood may come from the stomach in ulceration of that organ without any other symptoms. I

have found in autopsies active ulcers of the stomach, which during life never caused a symptom. A poor observer might tell us that he coughed up blood when in reality he had vomited it. When we are present during a haemorrhage we can generally decide very positively the source of the blood as to whether it comes from the lungs or the stomach.

Allow me to diagram the differential diagnosis in this way:

HAEMOPTYSIS.	HAEMATEMESIS. Usually later in life than haemoptysis, except in young women.
Generally previous pulmonary trouble.	Generally previous gastric trouble.
Blood coughed up.	Blood vomited after nausea.
Blood bright color, frothy, mixed with air.	Blood pure or mixed with food. Occasionally blood very dark, "coffee ground."
Blood-streaked expectoration after hæmorrhage.	None.
Only when blood swallowed. Usually absent.	Stools frequently black and tarry.
Haemorrhagic mass always alkaline.	Some of the mass gives an acid reaction.
Increase of bubbling rales in chest.	None.

This man had a slight cough between haemorrhages and for a year has had a marked morning cough and now we find the bacillus of tubercle in his sputa. The fact that his haemorrhages continued for over three and a half years, occurring almost every month is in no measure indicative of ulceration of the stomach but is strongly indicative of pulmonary trouble.

This man for a year has coughed up a great deal of white, tough, sticky mucus, with many whitish kernels. I have examined it twice for elastic fibres and have found them both times. Thisis positive evidence of a destructive process in the lungs and thus it usually is direct proof of tuberculosis. The sputa is boiled with sodium hydrate and the precipitate examined. The elastic fibres are seen as wavy fibres of some length with rather a definite alveolar arrangement. Those of you who own microscopes of only moderate power should make this examination. Their presence has a sure diagnostic significance.

The physical examination of this man's chest shows nothing marked. Impaired clearness of the percussion note is present over both apices. A prolonged rough expiratory murmur at these points seems to be the principal thing on auscultation. Allow me to impress upon you the advantage of always inquiring in auscultation in-

to the expiration separately from the inspiration. A rough prolonged expiratory murmur at the pulmonary apices means that the air has difficulty in getting out of the lung. Two factors detain it, either loss of elasticity of the air vesicles or obstruction in the bronchi- Elasticity of the air cells may be occasioned by overdistention as we find in emphysema or, and remember this, by deposits which impair their contractile power. If we had prolonged expiration at these apices and with it augmented clearness on percussion we would have to say that these apices were emphysematous, but our percussion note is impaired in its clearness. Such a condition is almost always caused by a tubercular deposit. This deposit not only gives us a prolonged expiration due to deficient elasticity of the air cells, but it transmits readily a portion of the bronchial sound to the ear and this sound is better and earlier perceived in expiration. The obstacle to the exit of the air may be entirely in the bronchial tubes. If the mucous membrane of the bronchi is swollen, prolonged expiration would be present. It is usually accompanied by mucous rales. An accurate study of expiration is of decided value and should be carefully studied. This patient has then slight tubercular trouble in both apices. The prognosis in his case is good.

* * *

Our next patient has had his present enemy within his citadel for over sixteen years. He first consulted me here in February, 1894.

J. P., 30, Russian, tailor, married, in Colorado since February, 1894, comes with a negative family history as regards tuberculosis. As a boy he was strong. Did not even have the diseases of childhood. At fourteen years of age he took a severe cold and his present trouble dates from that time. Ever since this he has had a cough. When he was twenty years of age he had his first haemorrhage. Since that time he has had forty or more. He had his last haemorrhage six months ago when he lost a full teacup of blood.

His present state is as follows: He feels weak and tired, has no pain in chest, coughs and expectorates both day and night, but most at night. He spits up much white and solid matter, all of which floats. For the past six months he has been very short of breath, with a poor appetite but bowels regular. At present he retains his usual weight, a hundred and fifty pounds. After his haemorrhage six months ago he had many night sweats, but has none now. The pulse is 126, the temperature is normal.

The physical examination shows the right chest considerably depressed in the upper half. He has an expansion of only one

inch. Over entire right lung anteriorly there is a marked rhonchal fremitus. To percussion the entire upper third of the right lung gives a high-pitched note. The sense of percussion resistance is marked. The extreme left apex gives a high pitched percussion note. Over left apex expiration and inspiration are accompanied by moist bubbling rales. This extends down to the fourth rib both anteriorly and posteriorly. The rest of this lung is normal.

Over the entire right lung we find mucous and submucous rales with an occasional crepitant rale at the apex and a few snoring rales at the base.

The heart and vocal bands are normal. The sputa contains a few bacilli of tubercle.

This man has been under continuous observation for over a year. An examination made an hour ago shows that the right lung has cleared up some, but that he now has more catarrhal involvement of the left lung. He has been working indoors for several weeks against my protest and to this is no doubt due his poor record at this time.

The most marked feature this man presents on examination is a rhonchal fremitus over the entire right lung anteriorly.

By a fremitus we mean certain tactile sensations conveyed to the surface of the chest. Ordinarily we speak of four varieties, (1) vocal fremitus, produced by the act of speaking or crying; (2) tussive fremitus, originated by coughing; (3) rhonchal fremitus due to the passage of air through bronchial tubes containing thick mucus or some other fluid. It is occasionally felt over cavities in the lungs; (4) friction fremitus, elicited by the rubbing together of roughened surfaces of the pleura.

When we apply our fingers to this man's chest while he is breathing quietly, we get a marked sense of a rolling sensation under our fingers over the entire right lung anteriorly. We recognize this to be something abnormal. We test his vocal and tussive fremitus and they are not increased. We apply our stethoscope and hear over the entire right lung mucous and submucous rales. The apex and upper one-third of the right lung gives us a high-pitched percussion note and an occasional crepitant rale with prolonged expiration. Our diagnosis for the right lung must be tubercular involvement of the upper third, bronchitis of lower two-thirds.

The left lung is found to be normal except that from the apex down to the fourth rib we have inspiration and expiration accompanied by moist bubbling rales. Persistent moist rales at an apex, in a case in which the bacillus of tubercle has been found in the sputa indicate a tubercular involvement in which softening has begun.

Let us briefly review this tubercular involvement. There was first deposited here, the tubercle, a mass of new cells. Others were scattered about it. Each tubercular nodule was surrounded by an area of tuberculous pneumonia. The areas were separated at first and the lung tissue between them was fairly healthy. By the deposition of tubercles and fresh pneumonia the solid areas merged one into the other. In the blood-vessels of the part an endarteritis occurred and the tuberculous areas became extra-vascular. They broke down by fatty degeneration or caseation as it is called. Such a process has gone on in this man's lung and all of it has been due to the presence of a vegetable fungus, the bacillus of tubercle.

* * *

M. J. F., 26, Canada, clerk, single, came to Colorado one year ago from Michigan, and came under our clinical care November 15th, 1893. His father died at the age of 73 of dropsy. His mother died of consumption at 55. One sister died of consumption. Three brothers died of some disease resembling malaria. He has one brother and one sister living.

Patient had no disease of childhood except measles. He had ague for one week in early life. Otherwise he enjoyed good health and was strong up to his present trouble.

This began in 1879. He took a severe cold and began coughing. For sixteen years this cough has never left him. For a long time it was dry and he would only occasionally bring up any sputa. In 1890 patient had his first pulmonary haemorrhage and soon after he had a second. He then enjoyed good health, except for a dry cough, until July, 1892, when within ten days he had four severe pulmonary haemorrhages. At this time he had night sweats. He then came to Colorado. Soon after coming he had four more haemorrhages. Since then he has enjoyed fairly good health.

At present he feels well, has some slight pain in the right chest anteriorly. Coughs most in the morning and on going to bed at night. Expectorates light colored, heavy sputa, which has been especially abundant since September, 1893. For two years he has been suffering with some shortness of breath. Appetite is good and bowels regular. No night sweating. Pulse 120, temperature 100. Present weight is 135 pounds, his former weight was 148. His voice was hoarse for three months but it is now clear. His chest is of fair size with some flattening of right chest anteriorly. The measurement for deep expiration is 32 inches, for deep inspiration 33½. Over the entire right lung there is increased vocal resonance and a high-pitched percussion note. The left lung gives

a clear normal percussion note. Over the right lung both anteriorly and posteriorly, mucous rales are heard both during inspiration and expiration and most of the lung gives a clear broncho-vesicular breath sound. Just below the right clavicle, a little to the left of the median line there is evidence of a cavity. The left lung is normal except for an occasional click at the extreme apex.

The heart sounds and area are normal. The vocal bands are relaxed and both move equally. They present no ulceration. The urine contains no albumen and no sugar. The sputa show no bacilli of tubercle.

We have to study in this patient but one lung, the right one. It will afford us much instruction however. The mucous rales heard so extensively indicate a bronchitis, the broncho-vesicular breath sounds with increased vocal fremitus and high-pitched percussion note indicate a consolidation of pulmonary tissue.

A distinctive characteristic of the normal vesicular murmur is its softness. That softness is wanting here. Instead we have a harsh breath sound, a union of the vesicular and bronchial sounds, a rough inspiratory sound devoid of softness with a prolonged somewhat blowing expiration. This we term, vesiculo-bronchial breathing. It exists in bronchitis, but with it in that disease we do not have a high-pitched percussion note as we have here. It also exists in diseases attended with compression of the lung tissue or with partial consolidation as we find in phthisis and pneumonia and as we find it here.

Just below his right clavicle I find evidence of a cavity. A pulmonary cavity is something that many medical men fail to recognize. In a recent case of my own on which I had a post-mortem, I had failed to diagnose a fair sized cavity at the left apex. The signs of a cavity are not always uniform, but by keeping in view certain strongly presumptive signs and not wandering off into the exceedingly fine distinctions of the books, we are not apt to go wrong many times.

Let me tell you why I diagnose a cavity the size of a hen's egg below this man's right clavicle. We have here a percussion note of almost tympanitic quality. Its pitch changes on having him open his mouth widely. On auscultation we get the so-called cavernous sounds, a blowing or tubular hollow sound, somewhat such a sound as we get over the trachea. This is never heard in this region without cause. With this blowing sound I find here coarse bubbling rales. They are not the mucous rales I hear over the rest of this lung. On his coughing we find these rales to have a certain metallic character. I have him count "five" and I find

the vocal resonance is greatly intensified. His voice comes to my ear as a great body of sound. On whispering, his voice seems to be conducted directly to my ear along the stethoscope, and the words uttered can be distinctly recognized. Nowhere else in this man's lung do these peculiar features exist. I can positively diagnose a cavity because of these signs.

* * *

Our next patient does not come to us because of his pulmonary state but because of symptoms referable somewhat to the pelvis of his left kidney. His pulmonary history I ascertained in taking my usual history of such a case.

Wm. B., 46; born in Missouri, married, came to Colorado in 1880. His father died at 77 of apoplexy. His mother died at 57, the cause of her death is not known. No consumption in any member of the family.

Patient had all of the diseases of childhood. Recovered from all perfectly. As a boy he was strong and well. At fifteen years of age he went to Austin, Texas, and in 1874 he took a position as a porter in an insane asylum there. He worked there until 1879. The indoor work and exposure caused him to cough. This began in the latter part of 1876. With the cough he had some expectoration and some falling off in weight. Late in 1876 he had his first pulmonary haemorrhage. Then his cough cleared up some and he got some better. For the next four years he had a cough most of the time and he had four or five haemorrhages. They were all very large ones and each left patient in a very weak state. He was never confined in bed, however, after the attacks. After one very severe haemorrhage, he came to Colorado. He denies syphilis or alcoholism.

Since coming to Colorado fifteen years ago, this patient has had five haemorrhages. The last was in the fall of 1882. After this haemorrhage he ate a full pint of salt, because his friends insisted upon his doing it and because salt had seemed to help him in other haemorrhages. Since this haemorrhage patient has had an occasional cough; a slight cold increases it markedly. For several month's during the past year he was perfectly free of any cough. For a month or two past he has had slight expectoration. He says he has been short of breath on exertion since 1882.

In August, 1894, he had cystitis, apparently idiopathic. He was in bed for several days. Since this he has had frequent urination both day and night. At present makes water four to five times at night. Examination of the urine shows nothing abnormal except crystals of calcium oxalate.

The examination of his lungs shows a magnificent chest. Under the right clavicle at the outer half there is just a bit of sinking. Expansion of chest is 4¾ inches. Percussion gives a marked high-pitched note over the upper one-third of the right lung. Palpation gives an increased vocal fremitus here. On auscultation the upper third of his lung gives prolonged expiration, vesiculo-bronchial breathing and an occasional submucous rale at extreme apex. The left apex down to the second rib, gives the same array of physical signs with a marked blowing cavernous sound.

Careful sounding of his bladder fails to find a stone. The finger inserted into the rectum shows the prostate glands slightly enlarged. The examination of his sputa shows no bacilli of tubercle.

In a word, our physical examination here shows a cavity at left apex with a fibroid tubercular involvement of right apex. One reviewing the pathology of chronic pulmonary involvement cannot fail to consider fibroid pneumonia, as it is a condition frequently met with. It is indeed nature's process of healing. It consists essentially in a gradual substitution to a greater or less extent of connective tissue for the normal lung substance. That has occurred in this man's right apex. If we should open his thoracic cavity the affected part would feel firm and fibrous; the visceral pleura would be found enormously thickened and firmly adherent to the costal layer. The involved lung area would feel like India rubber and at the point of normal lung tissue we would find some patches of compensatory emphysema. Very frequently we find, even in the oldest of cases, tubercles in the deeper layers of the pleura. A a rule the new connective tissue of the pleura as well as the new fibrous bands in the lung substance is considerably pigmented. With the contraction of the new scar tissue the vessels and bronchi appear to be crowded together and are dilated. We frequently find in such apices dilated bronchi which may be large enough at times to give us evidences of a cavity. The manner in which this new connective tissue is laid down is the same as in all interstitial inflammations. First an exudation of leucocytes takes place in the connective tissue, as the result of inflammation. Then new capillary loops from adjacent blood-vessels spring into the mass of exuded cells. Granulation tissue is now produced. The connective tissue cells of the part proliferate and new connective tissue bands are formed.

In all of the four cases reported, haemorrhages have been frequent and profuse. It is probable that but few cases of protracted pulmonary tuberculosis can be found in which haemoptysis is not a prominent symptom. There is good reason for this coming up of

blood. Usually it comes from eroded capillaries but not infrequently a considerable branch of the pulmonary artery either gives way or is perforated by erosion. Minute aneurisms are frequently present due to weakening of the arterial wall or to the vessel wall being drawn out by contraction of newly-formed connective tissue. The presence and the death of the tubercle nodule must destroy many capillaries. Haemorrhages must follow. The wonder is that every case of pulmonary tuberculosis does not have haemoptysis.

None of the cases reported present any dyspnoea except on exertion. This is a usual history in ordinary phthisis. The greater part of one lung may be diseased and much trouble may exist at the other apex without any shortness of breath. The probable explanation of this is that in phthisis there is always some anaemia and that the diminished lung space is sufficient to supply oxygen to the lessened number of red blood corpuscles.